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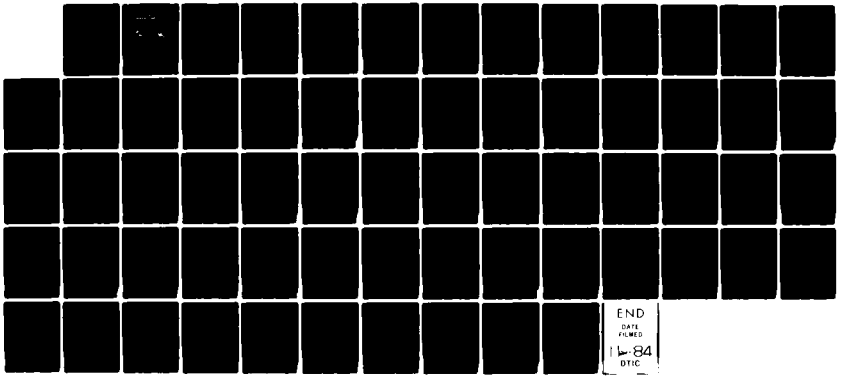

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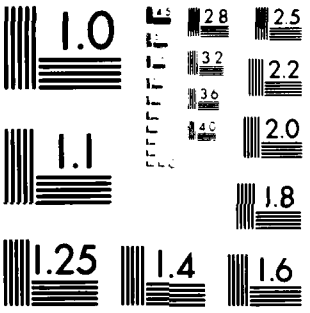

NAVAL MEDICAL RESEARCH INSTITUTE
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PATHOGENESIS AND TREATMENT OF CEREBRAL AIR EMBOLISM AND ASSOCIATED DISORDERS

P.W. Catron, J.M. Hallenbeck, E.T. Flynn, M.E. Bradley, and D.E. Evans

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A comprehensive discussion of the pathogenesis and treatment of cerebral air embolism and associated disorders is presented. Included are recommendations for diagnostic and therapeutic equipment and drugs to be available both inside and outside of the recompression chamber, and detailed rationales and schemata for administering therapy.

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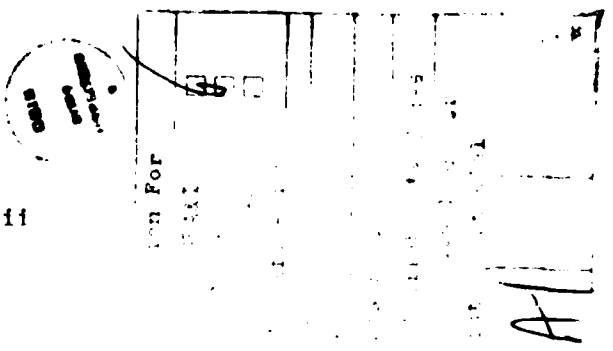

EXECUTIVE SUMMARY

This paper cites cerebral air embolism and associated disorders as an infrequent, but serious, problem in ascent training for submarine and diving personnel, and recommends methods for treating these disorders through equipment, drugs and supplies; staff training; and procedures.

The Mapleson "D" anesthesia bag-mask ventilator and a hyperbaric patient ventilator system are fixed chamber installations recommended to treat air embolism casualties. For temporary patient support, the Mapleson "D" ventilator is recommended because of its overboard dump system and ability to maintain positive end-expiratory pressure on the patient airway. For prolonged patient support, a hyperbaric patient ventilator system such as the Penlon Oxford Anesthesia ventilator is recommended because the operator controls ventilation, thus alleviating dangerously high levels of CO_2 in the patient at depth.

Extensive recommendations are made for portable equipment and supplies both inside and outside the recompression chamber. Those concerning outside equipment and supplies cover multiple areas: diagnosis; airway maintenence/control; cardiac resuscitation; IV fluids; blood drawing and major/minor line placement; monitoring; pleural drainage; and miscellaneous materials. Recommendations for drugs both inside and outside the chamber are similarly extensive and specify various forms of administration, such as preloaded disposable syringes, ampules, vials and tabs.

The area of staff training reveals that Navy diving training does not cover all skills necessary for expert care of critically ill embolism patients. It is thus the responsibility of the Escape Training Tank staff to ensure in-depth training for all staff personnel to treat these patients on scene. Major recommendations for in-depth training are as follows. All staff members

should be certified in Basic Life Support by an authorized instructor of the American Heart Association. All diving hospital corpsmen and diving medical officers should be certified in both Basic and Advanced Cardiac Life Support. Regular drills should be conducted to practice all patient care techniques. In addition, initial recompression techniques for patients with unstable cardiac function should be standardized and reviewed during practice drills.

Recommended treatment procedures for patients with air embolism focus on two categories: cases of primarily neurological injury and cases of cardiorespiratory arrest. Optimal treatment is detailed and differs for both categories of patients. In cases of neurological injury, the primary treatment for arterial air embolism is recompression to 165 feet of seawater (fsw). Ancillary therapy is cited, however, for patients who do not respond fully to standard treatment, e.g., those who improve at 165 fsw, but whose symptoms persist after 30 min, or for patients who develop sudden deterioration, such as a tension pneumothorax. Also recommended are specific measures useful in recompression chambers for patients with severe brain injury resulting from cerebral air embolism.

In cases of cardiorespiratory arrest, a specific protocol is suggested that represents a compromise between the dual treatment needs of recompression and defibrillation. The protocol modifies significantly the standard Treatment Table 6A because electrical defibrillation is currently deemed safe only at the surface.

In closing, it is recommended that patients who have suffered dysbaric arterial air embolism should receive post-treatment evaluation by neurologic examination; electroencephalogram; electrocardiogram and myocardial scanning; and pulmonary evaluation. In addition, recommendations are made for treating supplemental disorders resulting from extra-alveolar air, including mediastinal emphysema, subcutaneous emphysema, pneumopericardium and pneumothorax.

INTRODUCTION

This paper is intended to provide the current knowledge of the pathophysiological processes operant in pulmonary barotrauma and cerebral air embolism. Recommendations for staff training, therapeutic procedures, and medical equipment, drugs and supplies are presented. The discussions are deliberately very detailed, and are based on the authors' knowledge and experience of hyperbaric and critical care medicine.

BACKGROUND: STATEMENT OF PROBLEM

Individual ascent training, for both submarine and diving personnel, carries a small, but not insignificant, risk of pulmonary barotrauma, cerebral air embolism and death. In 1964 Moses, and in 1967 Van Genderen, reviewed the incidence of over-pressurization casualties and fatalities at the Escape Training Tank, New London Naval Submarine Base. The number of cases of gas embolism and fatalities that occurred with free ascent, bouyant ascent and Steinke Hood Training during the period 1947-1967 are presented in Table 1. The three victims of fatalities that occurred at New London during this period were noted to be pulseless before or during recompression. During the period 1977-1980 there were approximately 13,000 ascents of all types at the Escape Training Tank in New London. Four air embolisms occurred during this period, three as a result of free ascent training and one during Steinke Hood Training. There were no fatalities.

In 1977 Greene reviewed the experience of air embolism during training for the period 1954-1977 at the Submarine Escape Training Tank at HMS Dolphin. He estimated there had been 91 cases of air embolism with four fatalities out of a total 212,000 ascents. Two of the victims of fatalities, which occurred in the mid 1970s, were pulseless at the time of recompression and could not be revived. Greene's major recommendations included development of a safe and

Table 1. Air Embolism Occurrences at the Escape Training Tank, New London Naval Submarine Base, 1947-1967*

Dates	Method	Total Number of Runs/ Method	Number of Embolisms	Ratio of Air Embolism to # of Runs (%)	Number of Fatalities
1947-1967	Free Ascent	17,500	10	.057	2
1956-1966	Buoyant Ascent	132,000	14	.0106	1
1963-1966	Steinke	47,000	4	.007	0

*Compiled from Moses (38) and Van Genderen (54).

effective method of electrical defibrillation compatible with the environment of a wet hyperbaric chamber at 6 ATA of air, together with experimental studies to elucidate the consequences of cerebral arterial air embolism on the cardiovascular system.

As yet, no means of administering electrical defibrillation safely and effectively inside a hyperbaric chamber has been developed. It is unlikely that such a device will be available within the next several years. For the past five years, experimental studies conducted at the Naval Medical Research Institute have examined the cardiac effects of arterial air embolism. This work has clearly demonstrated that severe cardiac arrhythmias of neurogenic origin can occur as a result of cerebral air embolism. This research has also shown that these arrhythmias are accompanied by marked systemic hypertension. Therapeutic adjuncts have been developed to ameliorate these pathophysiologic processes.

In the past two decades there have been major advances in the development and implementation of effective cardiopulmonary resuscitative techniques in the United States. The successfulness of these techniques is unchallenged. It is felt, therefore, that the knowledge of the pathophysiological processes involving the circulatory system in cerebral air embolism and the application of up-to-date CPR procedures may decrease morbidity, as well as mortality, of cases of cerebral air embolism resulting from escape training.

The current situation at Escape Training Tanks appears to be less than optimal. The diagnostic and therapeutic equipment and medications available at these sites are in accordance with the requirements of the U.S. Navy Diving Manual. While much of what may be necessary to provide cardiopulmonary resuscitation and support a critically ill patient is included, there are significant omissions. These include the ability to obtain an

electrocardiogram, the ability to provide mechanical ventilation either inside or outside the chamber and the ability to defibrillate electrically outside the chamber. The training level of CPR of the Corpsmen and other personnel who would have to provide immediate resuscitative procedures appears to be less than desirable, as there is no requirement for these personnel to acquire either basic or advanced training in cardiopulmonary resuscitation.

PATHOPHYSIOLOGY OF CEREBRAL AIR EMBOLISM

Decompression Barotrauma

When an individual is exposed to a reduced environmental pressure, intra-alveolar gas expands, according to Boyle's law. In general, the tension on alveolar walls can reach critical levels if an individual has taken at least one breath of ambient air at raised environmental pressure before returning to the surface. Experiments in animals have demonstrated that intratracheal pressures of about 80 mm Hg (Polak and Adams, 1932) or transpulmonic pressures (defined as intratracheal minus intrapleural pressure) of 60-70 mm Hg (Schaeffer et al., 1958; Malhotra and Wright, 1960) are sufficient to rupture alveolar septa and allow gas to escape into interstitial spaces. The extra-alveolar gas may travel along perivascular sheaths and cause mediastinal emphysema and pneumothorax (Macklin and Macklin, 1944). It may also enter the pericardium, the retroperitoneum and subcutaneous tissues of the neck. When intrathoracic pressure drops during the first breath after pulmonary barotrauma, extra-alveolar gas can intravasate into torn vessels (Polak and Adams, 1932), migrate to the left side of the heart and enter the systemic circulation as particulate bubble emboli. Intratracheal pressures high enough to rupture alveoli can theoretically be reached by ascent from as shallow a depth as 4 ft (1.2 m) after full inspiration. Gas embolism has actually led to death following ascent from a depth of 7 ft (2 m).

Failure to exhale during ascent is one causal factor in the onset of pulmonary barotrauma and is prone to occur when a diver makes an emergency ascent in a state of panic. Some other explanation is necessary, however, to account for the observation that during submarine escape training, subjects have suffered embolism after ascents in which they exhaled as instructed. Air that has passed through a diseased bronchus and remained in a lung segment has been identified as the cause of embolism in two of these cases (Liebow et al., 1959). A broncholith of a tuberculous nature acted as a ball-valve in one case, and in another, the mechanism of bronchial obstruction was not determined. Occasionally, single or multiple cysts in a lung segment after rapid ascent suggest the possibility of an existing focal bronchial ball-valve as the means of obstruction (Collins, 1962).

Pulmonary compliance may be a factor related to the potential for pulmonary barotrauma. In relatively stiff lungs a nonuniform distribution of lung elasticity may increase susceptibility to barotrauma because the more compliant zones of the lungs are subjected to excessive strain. Some instances of alveolar rupture may arise from overly forceful attempts to exhale during rapid ascent through the water. During forced expiration at low lung volumes, the airways tend to narrow and act as check valves (Dayman, 1951). This tendency is exaggerated by immersion in water. Closure of the airways with air-trapping (mainly in basal lung regions) has been observed in human subjects exposed to head-out immersion (Dahlback and Lundgren, 1973). Such a finding led these investigators to postulate that vigorous exhalation at low lung volumes could actually predispose divers to air-trapping and contribute to alveolar rupture during rapid ascents.

In one series of 88 cases of pulmonary barotrauma, gas entered the torn pulmonary vessels and traveled into the systemic circulation, producing signs

and symptoms of involvement of the central nervous system (CNS) in roughly 75% of the sample (Elliott, Harrison, and Barnard, 1978). This high incidence of air intravasation may relate to the capacity for a compressed gas inhaled at depth to form a pressure gradient that facilitates its entry into torn vessels when it is trapped after rupturing alveoli. The actual incidence of gas intravasation and systemic arterial gas embolization may be even higher than suggested by conventional clinical monitoring for signs and symptoms. Electroencephalography, a more sensitive index of perturbation of the brain than patient history and physical examination under some circumstances, has disclosed evidence of cerebral gas embolism after submarine escape training ascents in the absence of any clinically discernible symptom or sign (Ingvar, Adolfson, and Lindemark, 1973).

Gas that enters the disrupted septal vessels migrates to the left atrium and the left ventricle via pulmonary veins and is ejected into the blood circulation as foamy particulates that distribute according to their relative buoyancy in blood (Van Allen, Hrdina, and Clark, 1929). In the head-up, erect position the brain receives the bulk of embolic air, while in a feet-up, inverted position the coronary vessels are primarily embolized.

In contrast to the neurological decompression sickness that occurs in subsaturation diving, the major target organ in dysbaric arterial gas embolism is the brain. Embolic gas lodges in arteries and arterioles, causing both distal ischemia (Waite et al., 1967) and endothelial damage at the site of obstruction (Broman et al., 1966; Philp, Inwood, and Warren, 1972; Johansson, 1978; Nishimoto et al., 1978). The active bubble surface may be responsible for immediate damage to the endothelium.

Within a few seconds of the embolism, cerebrospinal fluid pressure (CSFP) begins to rise and it remains elevated from several minutes to more than 1 hr,

depending on the volume of air entering intracranial vessels (De la Torre, Meredith, and Netsky, 1962). The rise in CSFP is probably due to vasoparalysis and extreme vasodilation of open intraparenchymal vessels. An associated increase in intracranial blood volume (Fritz and Hossman, 1979) also occurs.

One other aspect of intravascular air following pulmonary barotrauma is its apparent tendency to induce decompression sickness (DCS) after a decompression that would not be expected to produce DCS. Present evidence for this is inferential, but it appears that additional gas saturates the blood, which is already gas-laden. This leads to bubble growth and additional bubble formation sufficient to evoke symptoms of DCS. Cases involving an apparent concurrence of cerebral gas embolism and decompression sickness after no-decompression dives suggest the existence of this phenomenon.

One effect of recompression is the reduction of bubble size to a point at which perfusion pressure can drive the intravascular gas through the capillary bed and reestablish circulation. Recompression to simulated depths of 60-100 ft in a chamber has been observed to clear the air from pial vessels down to 30 μm in diameter (Waite et al., 1967). In cases of dysbaric gas embolism resulting from submarine escape training, recompression is instituted within minutes of the onset of symptoms. Case reviews demonstrate that gas embolism is curable in most cases, but that in 7-14% of cases the patient dies (Elliott, Harrison, and Barnard, 1978). In gas embolism associated with diving, the death rate is higher and neurological sequelae occur, most likely because of longer delays before recompression. Even after a delay of many hours, however, recompression may be associated with a dramatic resolution of symptoms (Mader and Hulet, 1979). Recent evidence (Branston et al., 1974; Heiss, Haykawa, and Waltz, 1976) indicates that protracted neurological dysfunction can occur without progressing to the irreversible state associated with infarction.

Although neuronal function recedes during the period that local blood flow is in the critical range of 12-20 ml/100 g/min, the nerve cells can survive in suspended animation within this range for prolonged periods. This state has been termed the "ischemic penumbra" (Astrup et al., 1977).

The therapeutic challenge of dysbaric gas embolism includes at least two major categories of problems (Hallenbeck and Greenbaum, 1977). One category includes immediate deaths due to apparent cardiorespiratory failure. Direct coronary embolization by gas may contribute to this catastrophe (Greene, 1977), but fascinating new evidence implicates neurogenic influences on the heart and lung, mediated by the autonomic nervous system and resulting from embolization of the brain stem (Evans et al., 1981). Another category involves secondary deterioration after an initial recovery from the embolic attack. Characteristically, a trainee breaks the surface after a training tower run and loses consciousness within a brief period. After one to several minutes he is urgently recompressed to a simulated depth of 165 ft (50 m) with air as the breathing medium. The trainee generally regains consciousness within 10 min of reaching this depth. He becomes alert, exhibits mental acuity, and shows no signs of neurological dysfunction when examined by a medical officer. Twenty minutes to two hours after this initial recovery, however, clinical deterioration may set in. If the initial episode of gas embolism involved a focal deficit, the same deficit may reappear. If loss of consciousness was the initial symptom, headache, confusion, blindness and seizures ensue during the recurrence. Since the deterioration often occurs during decompression from 165 ft (50 m), a potential contribution to the deterioration may be an obstructed flow of blood to the veins due to expanding mediastinal gas, or further embolization or release of vasoactive substances from damaged lungs. The deterioration sometimes occurs while the patient remains at 165 ft (50 m),

however. Other considerations might include increased intracranial pressure or formation of edema.

Any of these pathological events could contribute to a slowing of cerebral circulation, and work by Denny-Brown and Meyer (1957) and Osburne and Halsey (1975) suggests that in areas of acute ischemic brain damage, slow flows may eventually cease altogether. Recent work indicates that this process of progressive shutdown of nutrient flow could involve some sort of interaction between blood factors and elements in damaged vascular or neural tissue (Hallenbeck, 1977). A combination of parenteral pharmacological agents that is vasodilatory and prevents platelets from aggregating, and blocks the coagulation and prostaglandin systems (prostaglandin I_2, heparin and indomethacin) has been shown to eliminate impairment of reflow after global CNS ischemia and to promote neuronal recovery after focal ischemia induced by air embolism (Hallenbeck et al., 1982). This suggests that such a combination might be of benefit in cases of dysbaric cerebral air embolism complicated by secondary deterioration. Although their routine use is not indicated at present, therapeutic drugs validated in clinical studies of diseases with related pathophysiologies (e.g., acute embolic or thrombotic stroke) may be of future benefit in the therapy of air embolism that responds poorly to recompression therapy or leads to recurring symptoms of DCS during recompression.

Prevention of Arterial Air Embolism

Dysbaric arterial air embolism is best prevented by careful and thorough diver training, a comprehensive physical examination with special attention to the respiratory system for all diver candidates and rigorous adherence to established, safe diving procedures.

RECOMMENDED EQUIPMENT, DRUGS AND SUPPLIES

The following list of equipment, drugs and supplies will allow complete medical management of nearly all air embolism casualties. Advice concerning current NAVMEDCOM recommendations for major life-support items such as defibrillators and monitors may be obtained from NAVMEDCOM Code 42, (202) 653-1159.

Fixed Chamber Installations

A. BIBS Manifold and Overboard Dump System.

In addition to the standard 50-psig oxygen header, patient mask and overboard dump assembly, a quick-disconnect BIBS fitting should be provided to supply oxygen to an oxygen-powered resuscitator (e.g., Elder, Robertshaw) or a Mapleson "D" anesthesia bag-mask ventilator.

The oxygen-powered resuscitator is a small, simple device that can operate directly from the 50-psig over-ambient header. It permits ventilation of the unconscious patient by mask or endotracheal tube at the press of a button. Overpressurization of the airway is prevented by a spring-loaded relief valve. When consciousness is regained, the patient can continue to breathe from this system in a demand mode for a short time.

The Mapleson "D" bag-mask ventilator is also a simple device. It operates from a flow of oxygen delivered at ambient chamber pressure. For the inexperienced operator, this flow is best achieved by modifying a standard hospital 50-psig oxygen flowmeter to plug into the quick-disconnect BIBS fitting. For the experienced operator, only a needle valve connection to the BIBS is required. The Mapleson "D" has the advantage over simple self-inflating bag ventilators in that 100% oxygen and positive end-expiratory pressure (PEEP) can be delivered to the patient and the device can be readily used for controlled, assisted or limited spontaneous breathing. The device is

also adaptable to either a mask or an endotracheal tube. During resuscitation with a mask, the ability to maintain a variable amount of PEEP in the bag facilitates the maintenance of the airway by distending the hypopharynx. In the event of poor mask seal, ventilation can be maintained by increasing the flow of oxygen into the device. Connection to an overboard dump system is possible by changing the spring-loaded relief valve to one designed for scavenging of anesthetic gases (Fig. 1). In general, the Mapelson "D" is preferred over the oxygen-powered resuscitator because of the overboard dump and the ability to maintain PEEP on the airway.

Whenever possible, a second 50-psig header for mixed gas should be provided and should be plumbed so that the patient can be connected to either header without interruption of his gas supply. This second header allows for a variety of gas mixtures to be used for treatments at depths deeper than 60 fsw.

B. Ventilator

In general, neither the oxygen-powered resuscitator nor the Mapleson "D" are suitable for prolonged patient support. For the unconscious, intubated patient, both require continuous attention and divert physician resources from other aspects of the patient's care. For the spontaneously breathing patient, both may lead to arterial hypercapnia, especially in an environment where the ventilatory response to carbon dioxide is likely to be depressed. The problem with the oxygen-powered resuscitator is poor pressure-flow characteristics in the demand mode at depth. This may not be a problem for all devices. The problem with the Mapleson "D" is inherent in the design. Since the Mapleson "D" is essentially a flow-through rebreathing system, the inspired gas will contain an amount of carbon dioxide proportional to the patient's carbon dioxide output and the flow of fresh gas into the device. Since the incoming flow is limited, a large individual with a large CO_2 output may develop an

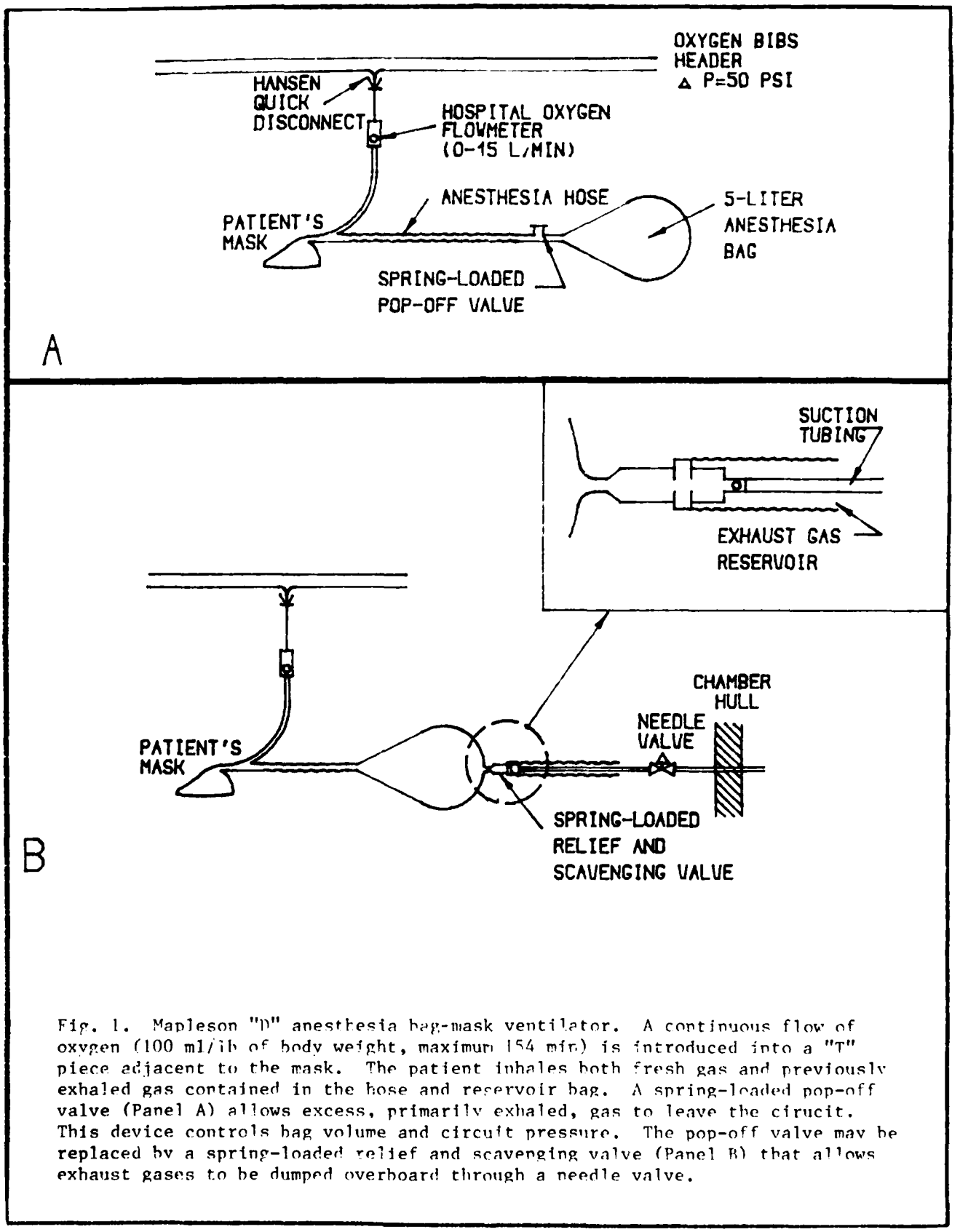

Fig. 1. Mapleson "D" anesthesia bag-mask ventilator. A continuous flow of oxygen (100 ml/lb of body weight, maximum 154 ml/kg) is introduced into a "T" piece adjacent to the mask. The patient inhales both fresh gas and previously exhaled gas contained in the hose and reservoir bag. A spring-loaded pop-off valve (Panel A) allows excess, primarily exhaled, gas to leave the circuit. This device controls bag volume and circuit pressure. The pop-off valve may be replaced by a spring-loaded relief and scavenging valve (Panel B) that allows exhaust gases to be dumped overboard through a needle valve.

unacceptably high level of inspired CO_2. This is not a problem with controlled ventilation because the operator can increase alveolar ventilation to compensate for the increased inspired CO_2.

For the reasons just enumerated, a volume-cycled ventilator with humidification, PEEP and intermittent mandatory ventilation (IMV) capability is highly desirable. In Royal Navy tests, the Penlon Oxford Anesthesia ventilator was found to perform satisfactorily in air to 6 ATA with minimal adjustment (Goad et al., 1981; Saywood et al., 1982). IMV capability can be added to this ventilator by attaching a 5-liter anesthesia bag to a "T" interposed in the inspiratory hose. The bag receives treatment gas directly from a 50-psig hospital-type oxygen flowmeter attached to the BIBS header. A one-way check valve in the "T" allows gas to flow from the bag to the inspiratory hose if the patient inspires spontaneously. During a machine breath, the check valve prevents the delivered tidal volume from entering the bag rather than the patient. The ventilator should be plumbed to deliver both 100% oxygen and mixed gas, so that intermittent oxygen-mixed gas exposures may be conducted. The exhaled gases should be directed overboard with an ambient pressure overboard dump system.

The overall configuration of the patient ventilation system used at NMRI is shown in Fig. 2.

C. Suction

Two 0 to 200-mm Hg hospital-type suction regulators and associated bottles should be mounted near the patient and plumbed to operate from the pressure differential between the inside and outside of the chamber. One regulator is for pleural suction, the other is for airway maintenance.

D. Patient Monitors

As a minimum, a 12-lead EKG should be hard-wired through the chamber

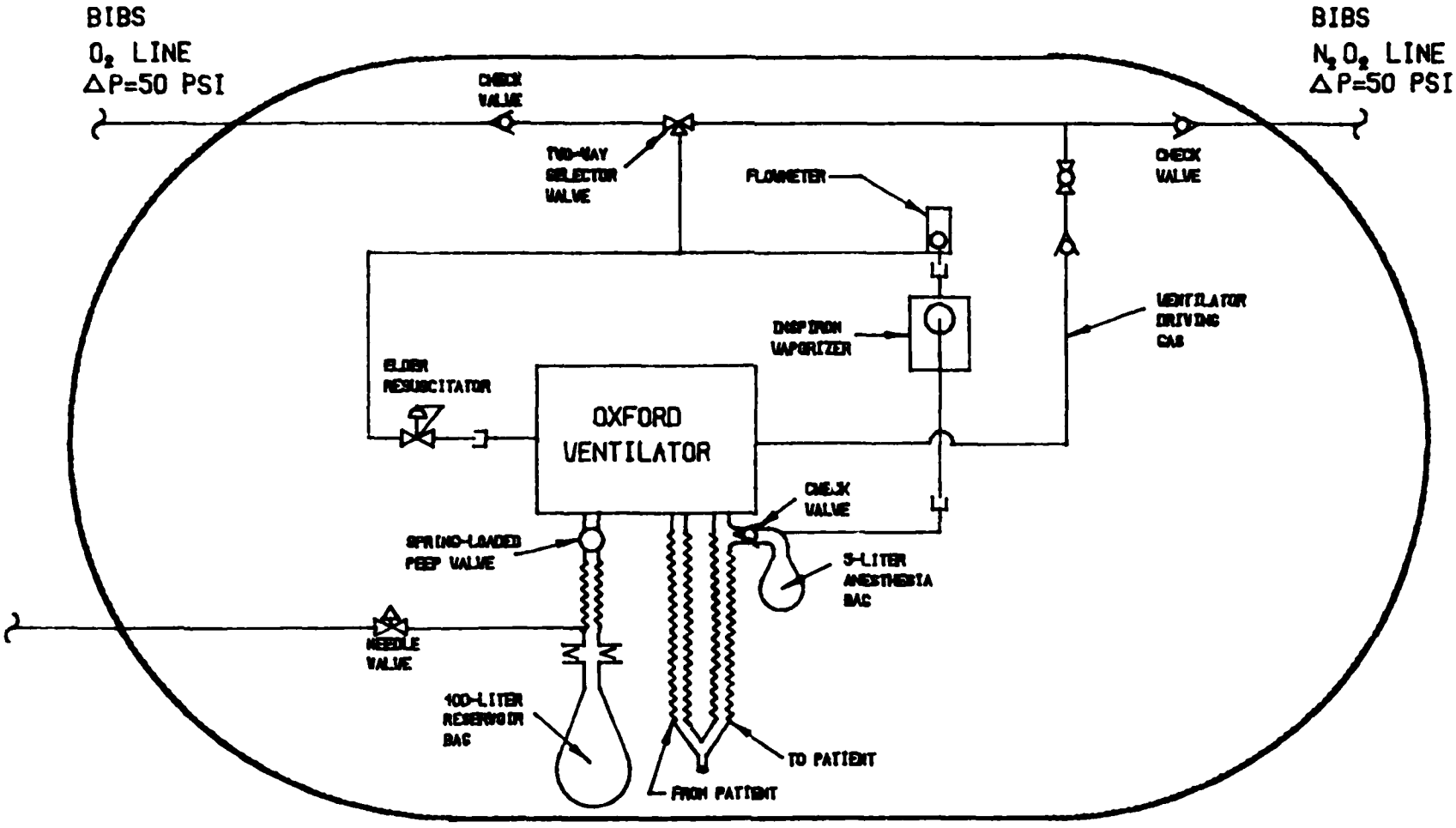

Fig. 2. Hyperbaric patient ventilator system. A two-way selector valve allows either oxygen or mixed gas at 50 psi over ambient pressure to be delivered simultaneously to an Elder resuscitator and a 0-15 L/min flowmeter. The Elder resuscitator, which in an emergency can be used directly on the patient, is plugged into the intake port of the Oxford ventilator to provide source of patient gas. The cam-actuated bellow of the Oxford ventilator delivers this gas to the patient through a standard anesthesia hose. An anesthesia bag on the inspiratory limb of the ventilator receives patient gas from the flowmeter and provides a gas source from which the patient can inhale between machine breaths. A check valve ensures that all machine breath flow to the patient rather than the bag. Exhaled gas passes through a spring-loaded PEEP valve and into a large reservoir bag. A needle valve allows this gas to be vented overboard, and double check valves prevent excessive suction or distension of the bag. The control circuitry of the ventilator is powered directly by the mixed gas header.

to an oscillographic monitor located outside. The inside chamber connector should be the same as that on the monitor so that the patient can be monitored either inside or outside the chamber without changing the lead harness. Additional hard-wired monitors that should be considered are arterial, venous and intracranial pressures, core body temperature, and thermodilution cardiac output. While some of these may require a specialty-trained physician for insertion, the availability of these monitors on-site insures that it is at least possible for the patient to receive intensive monitoring if needed. One monitor, arterial blood gases, is not likely to be practical in an operational chamber at present because of the amount of attention required. Blood drawn anaerobically and then decompressed, however, can be analyzed for CO_2 and pH outside the chamber with only small errors.

E. Oxygen and Carbon Analysis

In the event that saturation therapy may be required, the chamber should be equipped with atmospheric oxygen and carbon dioxide concentration monitors.

Portable Equipment and Supplies Located Inside Chamber

1. Stethoscope
2. Blood pressure cuff
3. Otoscope/Ophthalmoscope
4. Tuning fork set
5. Reflex hammer
6. Neurologic pinwheel
7. Penlight
8. Sinus transilluminator
9. Tongue blades
10. Cotton-tipped applicator

11. Disposable EKG electrodes

12. Laryngoscope (#3 Miller and MacIntosh blades)

13. Self-inflating bag-mask ventilator with medium adult mask*

14. #4 and #5 Geudel oral airways

15. #32 and #34F latex rubber nasal airways

16. 7.0 and 8.0-mm endotracheal tubes with stylet

17. 2-in waterproof tape

18. 5% lidocaine ointment

19. Yankauer nasopharyngeal suction tip

20. Penrose drain tourniquet

21. 16-g teflon IV catheters

22. 1000 cc Ringer's lactate or normal saline

23. IV infusion-set and extension tubing

24. 5, 10 and 20-cc syringes

25. Vacutainer tubes (red and purple top)

26. 18-g, 3 1/2-in intracardiac needle

27. 18-g and 21-g needles

28. Sealed isopropyl alcohol swabs

*Note: This device should be carefully selected as some models do not allow the operator to deliver an adequate tidal volume. The self-inflating bag-mask ventilator would be used in the event of oxygen system failure, precluding the use of the Mapleson "D" or the oxygen-powered resuscitator.

Portable Equipment and Supplies Located Outside Chamber

A. Diagnostic

1. Stethoscope

2. Blood pressure cuff

3. Otoscope/Ophthalmoscope

4. Reflex hammer
5. Tuning fork set
6. Neurologic pinwheel
7. Penlight
8. Cotton
9. Tongue blades
10. Alcohol thermometer
11. Sinus transilluminator
12. Ear syringe

B. Airway Maintenance/Control

1. #4 and #5 Guedel oral airways
2. Tongue blades
3. #32 and #34F latex rubber nasal airways
4. 5% lidocaine ointment
5. Cetacaine spray
6. 100%-oxygen "E" cylinder with 50-psig regulator
7. Mapleson "D" bag-mask ventilator (to operate from "E" cylinder and BIBS interchangeably)
8. Oxygen-powered resuscitation ventilator (to operate from "E" cylinder and BIBS interchangeably)
9. Portable suction device
10. Yankauer nasopharyngeal suction tips
11. Laryngoscope (#3 Miller and MacIntosh blades)
12. 7.0, 8.0 and 9.0-mm endotracheal tubes
13. Endotracheal tube stylet
14. Esophageal obturator airway
15. Tracheal suction catheters (14F)

16. Nasogastric suction tube and Tumee syringe

17. Tincture of Benzoin

18. 2-in waterproof tape

19. Precordial stethoscope

20. Esophageal stethoscope

21. Adult Magill forceps

22. Surgical lubricant

23. Small, medium and large adult anesthesia masks

24. Adult anesthesia mask retainer

25. Crycothyroidotomy kit

C. Cardiac Resuscitation

1. DC defibrillator with paddle EKG and synchronization mode

2. Electrode paste or disposable saline pads

3. Manual chest compressor

4. Transvenous pacing wire and portable pacemaker

5. 18-g, 3 1/2-in intracardiac needles

D. IV Fluids

Type	Size	Quantity
Ringer's lactate	1000 cc	3
D5/Ringer's lactate	1000 cc	1
0.9% NaCl	1000 cc	2
0.9% NaCl	250 cc	5
D5W	250 cc	2
D5 1/2 NS	1000 cc	2
Dextran 70	500 cc	1
Human serum albumin	100 cc	2

E. Equipment for Blood Drawing and Major/Minor Line Placement

1. Penrose drain tourniquet
2. Alcohol or betadine swabs; betadine spray; betadine ointment
3. 1% lidocaine for local anesthesia
4. 2.5, 5, 10 and 20-cc syringes
5. Vacutainer tubes (red and purple top)
6. 23, 21 and 18-g needles
7. 20, 18, 16, 14-g, 2 1/2-in teflon IV catheters
8. 14-g, 12-in intracaths
9. 14-g, 24-in intracaths
10. External jugular catheterization sets
11. Internal jugular catheterization sets
12. Swan Ganz introducer
13. #11 scalpel blades
14. Thermodilution Swan Ganz catheter
15. Sterile 2X2s
16. Sterile 4X4s
17. Sterile surgical towel pack (four towels)
18. Surgical gloves
19. 2-in waterproof tape
20. Macro-drip IV infusion sets
21. Micro-drip IV infusion sets
22. IV extension sets
23. Disposable plastic stopcocks
24. Calibrated pediatric drip chambers
25. Sorenson Dial-a-Flows®
26. Abbott pediatric "T" pieces

F. Monitoring

1. Disposable EKG electrodes
2. Disposable oral temp probes
3. Disposable CVP manometer
4. Rectal thermistor probe
5. Battery-operated thermistor probe box (if comprehensive monitoring not installed in chamber)
6. Arterial and venous pressure transducers
7. Intracranial pressure transducer
8. Pressure tubing; disposable, sterile transducer domes; disposable stopcock manifold
9. 12-lead EKG harness
10. Swan Ganz cardiac output harness
11. Urinary catheterization set
12. Urine collection/measuring device

G. Pleural Drainage

1. 10-cc syringes
2. 21-g, short-bevel, 1 1/2-in needles
3. 1% lidocaine for local anesthesia
4. 10-g argyle medicuts
5. 24F thoracic suction catheters
6. 24F trocar thoracic suction catheters
7. 10F trocar thoracic suction catheters
8. Sterile tube thoracostomy tray
   a. 1-Adsen Pick-up
   b. 2-curved Kelly hemostats
   c. 2-straight hemostats

d. 2-Rochester peans

e. 1-suture scissors

f. 1-needle driver

g. 2-Christmas trees

h. 1-10 cc glass syringe

i. 2-21 g, 1 1/4-in needles

j. 1-medicine glass

k. 20-4X4s

l. 1-#11 knife blade

m. 1-knife handle

n. 4-cloth towels

9. Heimlich valves

10. Disposable water-sealed thoracic suction units (e.g., Pleurovac)

11. Chest tube clamp

H. Other

1. Absorbent pads ("chucks")

2. Portapotty and replacement bags

3. Urinal

4. Bedpan

5. Sterile adult myringotomy kit

Drugs Located Inside Chamber (Preloaded Disposable Syringes)

Drugs	Unit	Number
Atropine	1 mg	2
Lidocaine	100 mg	2
Calcium chloride	1 g	2
Epinephrine (1/10,000)	1 mg	2
Sodium bicarbonate	44 meq	2

Drugs Located Outside Chamber

	Drugs	Suggested Quantity
1.	250-mg aminophylline ampules	4
2.	20-mg apresoline ampules	4
3.	1-mg atropine preloaded disposable syringes	4
4.	500-mg bretylium tosylate ampules	4
5.	1-g calcium chloride preloaded disposable syringes	4
6.	10-mg compazine ampules	4
7.	5 ml, 4-mg/ml dexamethasone multidose ampules	4
8.	10-mg diazepam preloaded disposable syringes	4
9.	0.5-mg digoxin ampules	4
10.	250-mg dilantin ampules	6
11.	50-mg diphenhydramine preloaded disposable syringes	4
12.	20-ml dobutamine vials (250 mg)	4
13.	200-mg dopamine ampules	4
14.	10-mg edrophonium ampules	4
15.	1-mg epinephrine ampules (1/1000)	4
16.	1-mg epinephrine preloaded disposable syringes (1/10,000)	4
17.	25-mg ephedrine ampules	4
18.	20-mg furosemide ampules	4
19.	50-cc 50% glucose ampule	1
20.	10-ml heparin multidose vials (1000 units/ml)	2
21.	250-mg hydrocortisone vials	2
22.	1-mg isoproterenol hydrochloride ampules	4
23.	20-meq KCL ampules	6
24.	4-mg levophed ampules	4
25.	1-g lidocaine vial (for IV infusion)	1

26. 100-mg lidocaine preloaded disposable syringes.....4

27. 12.5-gm mannitol ampules.....4

28. 500-mg methylprednisolone vials.....2

29. 5-cc 0.9% NaCl ampules.....25

30. 0.4-mg naloxone ampules.....4

31. 10-mg neosynephrine ampules.....4

32. 5-ml nitroglycerin ampules (5mg/ml).....4

33. 100 nitroglycerin tabs (1/200 gr).....1

34. 10-mg pancuronium bromide multidose ampules.....2

35. 1000-mg procainamide multidose ampules.....2

36. 1-mg propranolol ampules.....4

37. 44-meq sodium bicarbonate preloaded disposable syringes.....6

38. 50-mg sodium nitroprusside multidose ampules.....2

39. 200-mg succinylcholine multidose vials.....2

40. 1-mg terbutaline ampules.....2

41. 2-ml verapamil ampules (2.5 mg/ml).....4

RECOMMENDED STAFF TRAINING

The staff of the Escape Training Tanks will occasionally have to treat critically ill casualties resulting from escape training. The basic instruction provided by the Naval School of Diving and Salvage cannot be expected to encompass all the skills necessary for expert care of critically ill patients. It is the responsibility of the staff of the Escape Training Tanks to provide additional in-house training as necessary to ensure that its personnel are prepared to manage these patients on scene.

Plans for such additional training should take into account several factors:

(1) The staff will include fleet divers, diving hospital corpsmen and

diving medical officers, who will vary in their ability to take care of critically ill patients.

(2) Patients may well require advanced life support measures such as placement of peripheral and central intravenous catheters, endotracheal intubation and mechanical ventilation, defibrillation, chest tube insertion, and administration of emergency cardiac drugs.

(3) Diving medical officers will be readily available, but may not be on scene when an accident occurs. Therefore, fleet divers and diving hospital corpsmen should be trained to administer the initial medical care required by the victim.

(4) Serious casualties can be expected to occur infrequently and unpredictably. A regular program of drills will be required to keep patient care skills current.

With these factors in mind, the following recommendations are made for training:

(1) All members of the staff should be certified in Basic Life Support by an authorized instructor of the American Heart Association.

(2) All diving hospital corpsmen and diving medical officers should be certified in both Basic and Advanced Cardiac Life Support.

(3) Regular drills should be held to practice patient care techniques, including cardiopulmonary resuscitation (CPR), endotracheal intubation, line placement, Pleurovac® operation, defibrillation and administration of emergency cardiac drugs.

(4) Initial recompression strategies for patients with unstable cardiac function should be agreed upon in advance and reviewed during practice drills.

RECOMMENDED PROCEDURES

Patients with air embolism tend to fall into two categories: (1) those

with predominantly neurological injury and stable cardiovascular status, and (2) those who develop cardiorespiratory arrest. Optimal treatment differs for these two categories of patients.

Treatment Category 1: Neurologic Injury with Stable Cardiovascular Status

The primary treatment for arterial air embolism is recompression to 165 fsw on Treatment Table 6A. Recompression should be started at the earliest possible moment. Treatment Table 6A employs recompression to 165 fsw for 30 min, followed by ascent to 60 fsw over a span of 4 min, where 100% O_2 is breathed with intermittent air breaks for 1 hr. Two hour of oxygen breathing at 30 fsw completes the therapy.

The purpose of the initial recompression to 165 fsw is to mechanically reduce the size of arterial bubbles, and to promote the passage of bubbles through capillary beds. The rationale for oxygen breathing at 60 fsw is twofold: (1) to improve oxygen delivery to tissue, and (2) to reduce intracranial pressure. Improved oxygen delivery to tissue would be expected in view of the increased blood oxygen content due to dissolved oxygen approximately 4.5 volumes percent and the increased arterial PO_2, which provides a larger than normal driving force for diffusion of oxygen through tissue.

Several studies have shown that hyperbaric oxygenation lowers intracranial pressure (ICP). Miller and Ledingham (1969) demonstrated that breathing 100% O_2 at 2 ATA lowered ICP in dogs in which it had been raised by either inflation of an extradural balloon or cold injury to the brain. In the same study, they used an 85 Krypton washout technique to measure cerebral blood flow, and demonstrated that the reduction of ICP by hyperbaric oxygenation was due to an increase in cerebral vascular resistance, with a 20% decrease in cerebral blood flow. Furthermore, they showed that when autoregulation was completely lost --

as judged by the failure of cerebral blood flow to increase after inhalation of CO_2 -- hyperbaric oxygenation was ineffective in lowering intracranial pressure. Moody et al. (1970) and Pierce and Jacobson (1977) have confirmed the improved survival and neurologic outcome following hyperbaric oxygenation of dogs in whom head injury was simulated with an extradural balloon. In addition, the latter authors have reported reduction of ICP by oxygen breathing at 3 ATA in a patient with subarachnoid hemorrhage.

In the case of patients who are improving at 165 fsw, but whose symptoms have failed to clear after 30 min, several options exist:

1) Treatment may be continued at 165 fsw for up to a total bottom time of 2 hr. If available, a 40% oxygen-60% nitrogen mixture may be breathed by mask for 20-min intervals separated by 5-min air breaks. This mixture has a PO_2 of 2.4 ATA and offers many of the advantages expected of oxygen breathing at 60 fsw. If symptoms resolve or stabilize at the end of the 2-hr period, ascent is made to 60 fsw using the stops of U.S. Navy Treatment Table 4. At 60 fsw, continued ascent on Table 4, institution of an extended Treatment Table 6 or institution of air saturation may be elected, depending on the clinical status of the patient and the facilities available. Either of the latter two options is the preferred course of action. If after the oxygen breathing of an extended Table 6 at 60 fsw, the patient is well or has only minor residual symptoms, ascent is continued on Table 6. If significant damage persists, air saturation at 60 fsw may be desirable until the condition has stabilized. Oxygen may be administered periodically during this phase.

2) If symptoms are not completely resolved at 165 fsw at the end of 2 hr and additional time at depth appears to be of benefit, Royal Navy Treatment Table 72 (see Appendix) may be used. This table allows up to 4 hr of decompression at 165 fsw. Decompression is performed linearly rather than in

stages to prevent further pulmonary barotrauma. Approximately 15 hr are required to reach 60 fsw as opposed to 2 hr on Table 4. If available, hyperoxic nitrogen-oxygen mixtures ($PO_2 \approx 2.5$ ATA) may be breathed at depth and during the early phases of decompression to obtain the benefits of an increased PO_2. Care must be taken in the administration of these gases to avoid CNS oxygen toxicity. If significant residual damage exists normoxic nitrogen-oxygen saturation at 100 fsw or air saturation at 60 fsw may be considered.

For victims of cerebral air embolism who fail to improve significantly after 30 min at 165 fsw, a clinical judgment must be made whether symptoms represent continued obstruction of the cerebral circulation by air or the complex of cerebral edema, postischemic neuronal dysfunction and impaired reperfusion. If the injury is hyperacute (e.g., submarine escape training), the bubble mechanism cannot be discounted and additional recompression to 210 fsw (50 m) should be considered. Ascent can then be made on Royal Navy Treatment Table 71 (see Appendix). Hyperoxic gas mixtures may be breathed intermittently during decompression. Nitrox saturation at 100 fsw or air saturation at 60 fsw may be considered for significant residual damage.

If the injury is several hours old by the time treatment is initiated, bubbles in the cerebral circulation are probably of lesser importance than cerebral edema or impaired perfusion. In this case, direct ascent to 60 fsw and oxygen breathing on Table 6A are indicated. Air saturation at 60 fsw may be instituted if symptoms do not resolve. Alternatively, the patient may be given hyperoxic gas mixture at 165 fsw, followed by ascent to 60 fsw after 2 hr, as discussed earlier in option 1.

It is possible that ascent to 60 fsw on Table 6A will result in sudden deterioration of the patient. Such an abrupt deterioration should suggest

either the development of a tension pneumothorax or expansion of residual gas bubbles in the cerebral circulation. In either case, immediate therapy requires return to 165 fsw. If this is necessary, subsequent ascent to 60 fsw should follow USN Treatment Table 4 or Royal Navy Treatment Table 72.

Adjuvant Therapy

There has been considerable interest in recent years in the development of comprehensive protocols for the treatment of patients with various forms of severe brain injury (for reviews see Marsh, Marshall, and Shapiro, 1977; Hoff, 1978; Safar et al., 1978; Brennan, 1978; Bruce, Greenarelli, and Langfitt, 1978). These protocols require management of the patient in an intensive care unit. It is obvious that some of these specialized techniques, e.g., intracranial pressure monitoring, are not feasible for use in recompression chambers. In addition, the degree to which those measures should be extrapolated to victims of cerebral air embolism may be questioned. It seems reasonable, however, that some of the measures currently advocated for treatment of severe brain injuries should be useful in the treatment of patients with air embolism until such time that optimum adjuvant therapy has been determined experimentally. Specific measures that should be considered include:

1) Steroids

A large intravenous bolus of dexamethasone (1.0-1.5 mg/kg) should be considered for treatment of cerebral edema. If the patient responds completely to recompression and experiences no further deterioration, no additional steroid therapy is recommended. If clinical deterioration warrants continued therapy, additional doses of dexamethasone (0.2 mg/kg) may be given at 6-hr intervals. The dosages given here are those recommended by Safar et al. (1978) for treatment of global ischemia; they are large initial doses. The optimal

dose for treatment of cerebral edema is uncertain. The study by Faupel et al. (1977) of patients with head injuries demonstrated that low-dose dexamethasone therapy (12 mg IV, then 4 mg IM every 10 hr) was useful, but greater benefit was seen in the group that received high-dose therapy (100 mg dexamethasone IV initially, 100 mg IM in 6 hr, then 4 mg IM every 6 hr). The extent to which such high dosages will increase the frequency of complications from steroid therapy is uncertain. Other steroids or combinations of steroids have been suggested on theoretical grounds; reports of their use in air embolism in humans are not available to evaluate their respective values.

2) Fluids

Decisions made regarding fluid therapy must take into account the probable coexistence of three problems: vasogenic edema, cytotoxic edema and impaired reflow. The first two conditions are most likely to respond to restriction of fluids to one-half to two-thirds of maintenance values. These fluids should be crystalloid in nature, should contain glucose to provide substrate to the brain (Safar et al., 1978) and should contain normal daily maintenance quantities of electrolytes. A rise in plasma osmolality and hematocrit may be expected. A rise in the latter will have a negative impact on cerebral reperfusion because of the increase in blood viscosity. Further research is needed to determine whether fluid restriction or hemodilution is best when air embolism occurs.

3) Hemodynamic Support

Hypotension, if present, should be corrected promptly to restore cerebral blood flow. If pressor agents are required, dopamine or epinephrine are reasonable choices. Dopamine in doses of 1-10μmg/kg/min increases coronary blood flow, renal blood flow and cardiac output. At these dosages, myocardial consumption of 0_2 does not increase (Goldbery, 1974). If

hypotension can be corrected with doses in this range, dopamine is preferred to epinephrine. At higher dosages, dopamine increases peripheral vascular resistance and myocardial consumption of O_2 and decreases renal flow. It has few advantages over epinephrine.

The effects of pressor agents on cerebral hemodynamics after air embolism are not readily predictable, and may depend upon the status of the blood-brain barrier. When the blood-brain barrier is intact, epinephrine is said to have little direct effect on cerebral vascular resistance (Sokolof, 1959). The effect of norepinephrine depends on the level of systemic blood pressure prior to infusion: if the patient is normotensive prior to infusion, norepinephrine increases cerebrovascular resistance and lowers CBF; if hypotension exists prior to infusion, the net effect of norepinephrine is to increase CBF (Sokolof, 1959). The effect of dopamine in this situation is disputed. Von Essen (1972) reported reduction of CBF with low-dose infusion of dopamine in the dog, and increased CBF with high-dose infusion, but these findings have been questioned by McCulloch and Harper (1977).

It has been suggested that in the presence of an altered blood-brain barrier, systematically administered monoamines may gain access to the cerebral interstitium and directly influence cerebral metabolism (McCulloch and Harper, 1977). In this instance, alterations in cerebral blood flow may be secondary to changes in cerebral metabolic rates.

Deliberate induction of hypertension has been proposed as a measure to promote cerebral reflow. Wise, Sutter, and Barkholder (1972) reported improvement of neurologic deficits with hypertensive therapy in five of 13 patients suffering from focal brain ischemia. Data from animal models of global ischemia suggest that artificial hypertension may worsen neurologic deficits by increasing vasogenic edema and intracranial pressure (Bleyaert et

al., 1978). The usefulness of artificial hypertension in cerebral air embolism is unknown. Because evidence for the benefits of hypertensive therapy is conflicting, and because required accurate monitoring of blood pressure is not feasible in most recompression chambers, this measure is not recommended.

4) Position

A supine position should be adopted during the initial stages of therapy. Once therapy is underway in a recompression chamber and blood pressure stabilizes, a moderate elevation of the head may be preferred to reduce cerebral venous pressure.

5) Ventilation

Problems connected with ventilation and protection of the lungs from aspiration are likely to be among the most difficult aspects of the management of patients in the recompression chamber. Obviously, it is important to provide adequate oxygenation. Hypercapnia can be expected to markedly elevate ICP in a patient with vasogenic cerebral edema and must be avoided. Since objective indices of adequate ventilation, such as end-tidal or arterial PO_2, will not be available in most recompression chambers, the physician must rely on clinical judgment to determine the adequacy of ventilation. Clinical assessment should be made of the adequacy of both the patient's airway and his level of ventilatory effort. Insertion of a nasal airway and manual extension of the mandible may be useful if the adequacy of the patient's airway is questionable. A stuporous, combative patient may not tolerate these measures. If this is the case, i.e., if these measures are insufficient to maintain the patient's airway, or if protective reflexes of the airway are absent, endotracheal intubation will be required.

Intubation should be performed only when indicated clinically and not as a prophylactic measure. Indications include inadequate ventilation and the

absence of protective reflexes of the airway. The only situation where intubation would be avoided in escape training if one or both indications were present would be the casualty whose circulation is intact. The expectation in this situation is for prompt recovery of consciousness once recompression begins.

Even in the most ideal circumstances, intubation of a patient with raised intracranial pressure can be extremely hazardous. If intubation of such a patient is not done skillfully, the patient may struggle during the procedures, which will raise cerebral venous pressure. In addition, airway reflexes may cause marked elevations in arterial blood pressure. If the patient's ICP is elevated to begin with, these two effects may further elevate it to the point that herniation occurs. It is obvious that these difficulties are only compounded in a recompression chamber. Therefore, if the need for intubation is anticipated, the assistance of an anesthesiologist should be obtained. If this is impossible, then intubation should be performed as smoothly as possible.

Two options are available for intubation:

(a) Blind nasotracheal intubation is the preferred method for the inexperienced, unless the patient is flaccid and has no airway reflexes. In the latter case, oral intubation is preferred. Blind nasotracheal intubation may be performed after topical anesthesia of the nose and oropharynx with cocaine and cetacaine and/or lidocaine. Anesthesia of the hypopharynx and larynx are to be avoided. This procedure has the important advantage of leaving intact the patient's spontaneous ventilatory effort and protective reflexes of the airway during intubation. The nasotracheal tube may be easier to place than an orotracheal tube, and once in place is usually better tolerated by patients than an orotracheal tube. The technique is reasonably

simple. After adequate anesthesia is achieved, a #34F latex rubber nasal airway is lubricated with sterile jelly and passed through the intended nostril to ensure patency and provide some dilation. This is then removed and a lubricated #8 endotracheal tube is passed through the same nostril, while care is taken to hug the floor of the nasopharynx. A pop and a sudden increase in the ease of passage will be felt as the tube enters the oropharynx. The physician then listens to the intensity of the breathing sounds as he directs the tube toward and into the larynx. Failure to pass the tube usually indicates it is either off to one side or posterior to the larynx. The tip of the tube may be directed side to side by twisting the end sticking out of the nose. If the tube is posterior, extension of the head or the neck often corrects the situation. It is often possible in difficult cases to expose the posterior pharynx or even the larynx with a laryngoscope and pass the tube under direct vision. Magill forceps may be helpful in this situation. Unless the patient is severely obtunded, some cough will be encountered when the tube crosses the vocal cords. This is unavoidable, but may be minimized by prior injection of 100 mg of lidocaine IV.

(b) The alternative approach is to intubate the patient orally. The advantages of this method for the trained anesthesiologist are that hypertensive reflexes can be minimized by prior administration of lidocaine and pentobarbital, and struggling can be prevented with short-acting muscle relaxants. The patient's own efforts at ventilation are abolished, however, necessitating prompt placement of the tube. This procedure must be reserved only for those skilled at intubation and ventilation of unconscious individuals.

6) Hyperventilation

If the patient is intubated, hyperventilation should be induced. The

aim of hyperventilation is to lower ICP by reducing cerebral blood flow and blood volume. Modest hyperventilation to 25 mm Hg of $PaCO_2$ is generally recommended. Reduction of $PaCO_2$ below this level may be deleterious because of excessive lowering of cerebral blood flow and fall in cardiac output (Safar et al., 1978). In some centers, PO_2 of the jugular venous bulb is used to determine this cutoff point, but these methods are not applicable to present-day chambers. Because blood gases will usually not be available, some measures of respiratory minute ventilation must be used in order to avoid excessive hyperventilation.

The possible benefits of hyperventilation in reversing intracranial steal are speculative. Soloway et al. (1969) demonstrated the value of hyperventilation in reducing infarct size in the dog if it was started prior to occlusion of the middle cerebral artery. A subsequent study, however, showed no effect if hyperventilation was started 1 hr after occlusion (Soloway et al., 1971).

Positive pressure ventilation, given manually or with a respirator, may cause additional pulmonary barotrauma in these patients. If, however, the patient's ventilation is inadequate, this risk must be accepted. PEEP should be avoided unless oxygenation deteriorates.

7) Sedative and Anticonvulsant Medication

(a) Intubated Patients

Most intubated patients who are not deeply comatose require some sedation. They may also require medication for seizures. All sedative and most anticonvulsant medications depress ventilation, blood pressure and level of consciousness to some extent. Deep depression of the patient necessitates total control of ventilation and may require pressor drugs to correct iatrogenically-induced hypotension. This situation is not desirable for

recompression chamber therapy. If sedative and anticonvulsant drugs are necessary, the dosages chosen should be ones that produce the desired effect, but cause the smallest possible depression of the patient. Options follow.

(1) Barbiturates

Barbiturates provide anticonvulsant protection as well as sedation. In addition, barbiturate anesthesia may protect the brain from hypoxic injury. In animal studies barbiturates have been shown to be protective in both focal and global ischemias (Smith, 1977). The mechanism of this action is uncertain but may include reduction of ICP, free-radical scavenging, reduction of cerebral metabolism or other effects (Safar et al., 1978). The optimum dose of barbiturates is controversial. Most studies in which a protective effect has been noted have employed relatively large doses with deep anesthesia. Deep barbiturate anesthesia may cause circulatory depression and should not be used in the recompression chambers. Circulatory depression is especially likely if volume depletion is present. Barbiturates do not provide good airway anesthesia. Attempts to prevent a patient from bucking against an endotracheal tube by giving repeated boluses of barbiturates will not correct the situation until a very high and undesirable dose is obtained.

A reasonable starting dose of Thiopental is 100 mg IV. If the patient requires more anticonvulsant medication as treatment is continued, additional boluses may be tried -- up to a total dose of 4 mg/kg -- provided that hypotension is not a problem.

(2) Valium

Valium may be useful as general sedation or for seizure control. Valium may be given in 5-mg IV boluses up to a total dose of 20-30 mg.

(3) Morphine

Intravenous morphine in boluses of 2 to 5 mg IV is very useful for supressing bucking against an endotracheal tube. Hypotension may occur with its use, especially if the patient's circulatory volume is depleted.

(4) Dilantin

The major disadvantage to dilantin is the difficulty of obtaining satisfactory blood levels in a short period of time. Approximately 1 gm of dilantin is necessary as a loading dose and is usually given orally over one to two days. Dilantin may produce severe hypotension, atrioventricular (A-V) block, asystole or ventricular fibrillation when administered by rapid intravenous injection. For this reason, dilantin has limited usefulness for recompression chamber therapy.

(5) Muscle Relaxants

Because of the possibility of accidental extubation and difficulties associated with reintubating patients in a recompression setting, muscle relaxants should not be used.

(b) Nonintubated patients

Sedation should be avoided in patients who are not intubated. If anticonvulsants are necessary, IV Valium is generally the drug of first choice. If Valium fails to control seizures, barbiturates may be tried. These drugs may suppress ventilation and necessitate intubation.

8) Osmotherapy

Hypertonic solutions, i.e., urea or mannitol, temporarily lower ICP by shrinking normal brain tissue, but may be associated with rebound increases in ICP (Fishman, 1975). Their use in most clinical situations is as a temporizing measure, pending definitive intervention such as surgery. These agents should probably not be used routinely for victims of air embolism. If deterioration

is marked, however, and impending herniation is suspected clinically, mannitol may be given by rapid intravenous infusion in doses of 0.5-1.0 mg/kg.

9) Promotion of Reflow

The role of agents intended to restore cerebral blood flow (dextran, heparin, sodium nitroprusside, prostacyclin) is uncertain at the present time. Until data are available demonstrating their usefulness, these agents are not recommended for routine use.

Category 2: Cardiorespiratory Arrest

Pathogensis

The studies of Evans, Hardenberg, and Hallenbeck (1977) in which air was infused into the left ventricle of animals during mechanical ventilation suggests that cardiac arrest may be produced by one of two mechanisms.

The first of these mechanisms is gaseous embolization of the coronary arteries with subsequent myocardial ischemia, infarction and cardiac arrest. Experimental animals in this group developed hypotension, depression of left ventricular contractile force, EKG evidence of myocardial ischemia or injury, and subsequently died.

The second mechanism involves embolization of the posterior cerebral circulation with massive hypertension, a high rise in plasma cathecholamine levels and marked ventricular arrhythmias. These arrhythmias may be prevented by prophylactic administration of autonomic blocking drugs, but the hypertensive response cannot be blocked by these drugs or by cervical cord section. The latter observation suggests that the hypertension results from a substance elaborated in the brain, perhaps angiotensin. Interestingly, preliminary experiments indicate that lidocaine administered prophylactically in doses of 5 mg/kg and greater can attenuate the entire response in a

dose-dependent manner. Lidocaine also prevents the rise in ICP that is seen in this situation.

Treatment Category 2: Cardiorespiratory Arrest

A proposed treatment protocol is shown in Fig. 3. The rationale for various aspects of the protocol is discussed below. (The numbered paragraphs refer to numbers shown in Fig. 3.)

The protocol shown in Fig. 3 involves considerable modification of Table 6A. Modifications of this sort are suggested because electrical defibrillation is deemed safe for use only at the surface. If a defibrillator safe for use in recompression chambers can be developed, then all aspects of resuscitation should be possible within the chamber and could be performed while following Treatment Table 6A.

The protocol of Fig. 3 should not be regarded as inviolate. It is suggested as a means to compromise between the need to recompress and the need to defibrillate.

The diagnosis of apnea and cardiac arrest is made by inspection and by rapid palpation of the carotid pulse. Ventilation and manual chest compression are begun immediately and continued until an adequate blood pressure is maintained spontaneously.

(1) Endotracheal intubation is the preferred technique for ultimately securing the airway. Due to the relatively cramped quarters of most recompression chambers, intubation is likely to be very difficult when attempted at depth. Therefore, unless the medical officer is confident of his ability to intubate the patient in the chamber, a short delay at the surface for this purpose may be justified. If an endotracheal tube cannot be passed, an esophageal obturator airway may be useful. If such an airway is used, the newer models, which permit gastric suctioning via a nasogastric (NG) tube, are

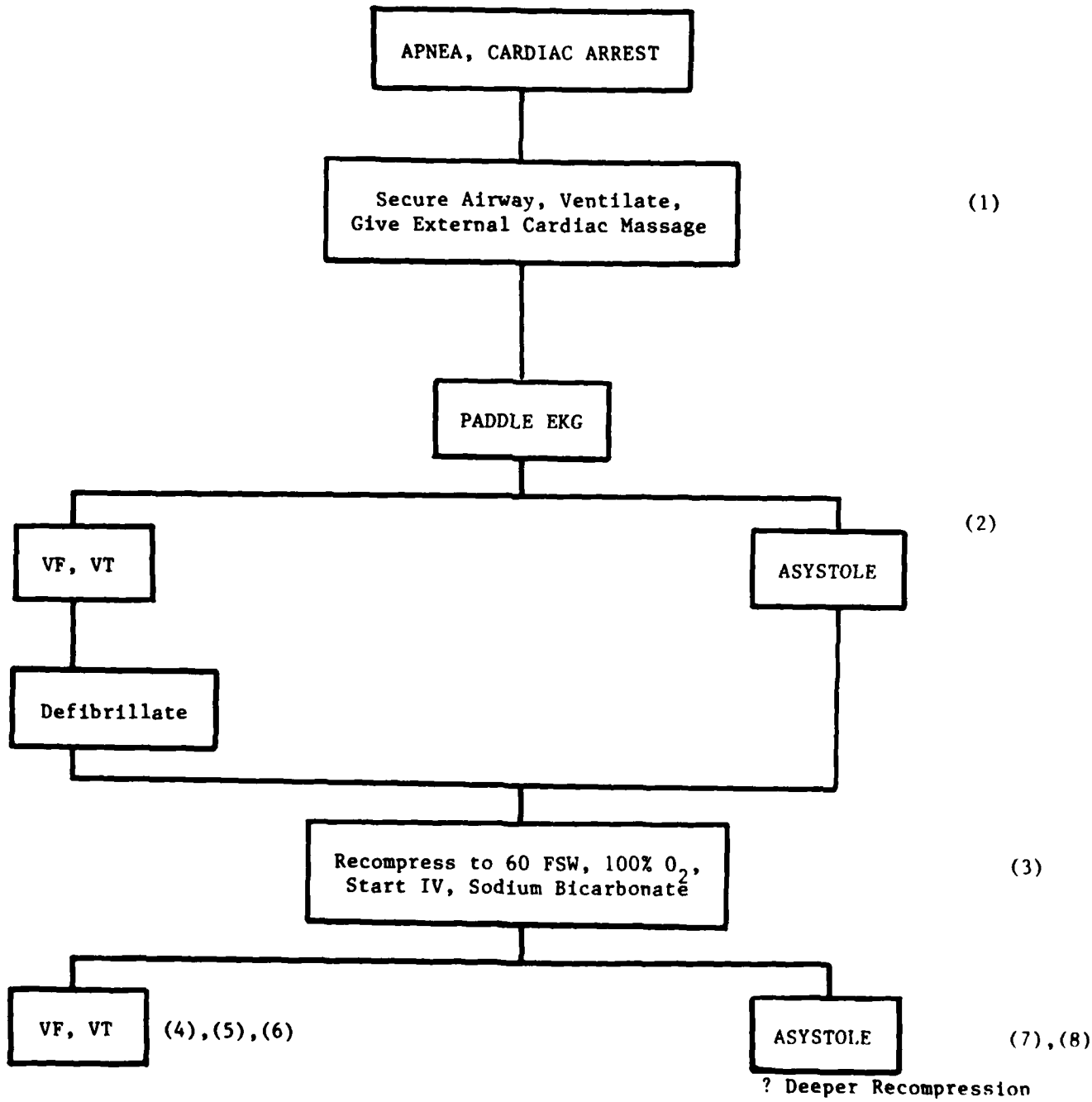

Fig. 3. Algorithm for management of victims of air embolism who present with cardiac arrest. Numbers in the figure refer to comments made in the text. This algorithm is intended only as a guide to management of these patients.

preferred and may lessen the severity of the regurgitation that occurs after removal of the airway.

(2) Time should not be taken at the surface to connect standard EKG leads. The initial diagnosis of the patient's arrhythmia should be made with a defibrillator that is capable of sensing the EKG through the paddles. If only a standard defibrillator is available, one attempt to defibrillate the patient prior to recompression should be made. This attempt should be made only if the patient is dry in order to minimize the danger of electrical shock to the rescuers. In the escape training setting, patients will most often be wet. In this case, time should not be lost drying and defibrillating the patient. Prompt recompression to 60 fsw is preferred. If a paddle EKG demonstrates asystole, the patient should be recompressed to 60 fsw with no further delay.

(3) Initial recompression to 60 fsw rather than 165 fsw is recommended primarily because of the probable need for defibrillation. At the present time, defibrillators are considered unsafe for use in recompression chambers because of the risk of electrical shock and chamber fire. Most patients with ventricular fibrillation or ventricular tachycardia (potentially reversible rhythms), however, require defibrillation in order to restore normal sinus rhythm. Initial recompression to 60 fsw and ventilation with 100% O_2 is a reasonable compromise. The recompression and oxygen breathing should promote rapid resolution of gas bubbles in the circulation and allow prompt ascent to the surface for defibrillation at any time within the first 60 min with minimal risk of decompression sickness for the tenders or the patient.

At 60 ft an IV should be started and sodium bicarbonate given. Because of the need for prompt recompression, it is preferrable to start the IV at treatment depth rather than delay at the surface.

(4) Standard EKG leads should be attached to 60 ft and the patient's arrhythmia diagnosed.

If ventricular fibrillation is present, therapy with bretylium tosylate should be considered. Bretylium has been shown to be effective in the treatment of recurrent ventricular arrhythmias that are resistant to drugs (Heissenbuttel and Bigger, 1979). In addition, bretylium may be useful for converting ventricular fibrillation to normal sinus rhythm (NSR) without the use of countershock. In one uncontrolled series in humans, bretylium was used successfully to chemically defibrillate six episodes of ventricular fibrillation in five patients (Sanna and Arcidiacono, 1973). Two patients of this series did not reach NSR with bretylium, but achieved a stable supraventricular rhythm after one countershock.

The optimal role for bretylium in a cardiac arrest situation has not been defined. In particular, the interaction of bretylium with other antiarrhythmic drugs and the hemodynamic effects of bretylium are not completely understood at present. No other known agent, however, offers a potential for chemical defibrillation. Successful chemical defibrillation with bretylium would obviate the need to surface the chamber in order to apply decompression countershock.

Bretylium, if administered, should be given intravenously. The currently recommended bolus dose is 5 mg/kg (JAMA, 1980). This dose may be repeated. Cumulative doses should not exceed 30 mg/kg. Bretylium may also be administered as a continuous intravenous infusion at a rate of 1-2 mg/min.

If the patient's initial rhythm is ventricular tachycardia, lidocaine or bretylium should be given. Lidocaine dosage is 1 mg/kg IV bolus. Lidocaine boluses may be repeated, but the maximum cumulative bolus dose should not exceed 300 mg. Lidocaine may also be given as a continuous infusion at rates

of 1-4 mg/min. When bretylium is used to treat ventricular tachycardia, the recommended dosage is 5-10 mg/kg diluted in 50-100 ml 5% dextrose in water (D_5W) or normal saline (NS) and infused rapidly. In view of the findings of Evans, Hardenberg, and Hallenbeck (1977) discussed earlier, lidocaine may have a specific beneficial effect in this situation.

(5) If drug therapy is unsuccessful and a shockable rhythm persists, the chamber should be surfaced and ventilated, and the patient should be defibrillated electrically. If the patient and chamber are wet, it may be advisable to remove the patient from the chamber and dry him prior to defibrillation. Unless NSR is restored promptly, the medical officer must make a decision at this point between continuing drug therapy and defibrillation at the surface or recompressing to 60 fsw and continuing drug therapy at depth. If the initial period of recompression was 30 min or longer and if a shockable rhythm persists, continuation of therapy with drugs and defibrillation at the surface for a short period is probably reasonable. Subsequent drug therapy at this point might include additional boluses of bicarbonate, epinephrine, lidocaine, procainamide or bretylium. If these measures are unsuccessful, intravenous propranolol should be tried because heightened sympathetic tone secondary to inadequate circulation in the posterior cerebral area or exogenous cathecholamines may be the mechanism contributing to the arrhythmia.

(6) At this point in the resuscitation, additional therapy will depend upon the patient's clinical course. If the patient's ventricular arrhythmia has degenerated into asystole, he should be recompressed to 60 fsw for further drug therapy. Again, the rationale for choosing 60 fsw as the recompression depth is the anticipated need for defibrillation after additional drug therapy. <u>All subsequent decisions to surface for defibrillation must take into account the tenders' requirements for decompression</u>.

(7) Therapy for asystole will depend in large part upon the medications the patient has received up to this point. Sodium bicarbonate, epinephrine, isoproterenol and calcium chloride are standard drugs in the therapy of asystole. Because of the possibility that vagal reflexes may be involved, atropine should also be given.

(8) If asystole persists despite drug therapy, deeper recompression to 165 fsw should be considered. The decision to recompress to 165 fsw rather than remain at 60 fsw can only be made on scene. The medical officer must weigh the need for deeper recompression for treatment of neurological injury against the need to be near the surface in order to defibrillate the patient. If recompression to 165 fsw is chosen, drug therapy, ventilation with chamber atmosphere and external cardiac message should be continued.

If treatment at 165 fsw for asystole is continued for more than 30 min, subsequent decompression to 60 fsw must be made from Table 4. From 60 fsw to the surface, decompression for both patient and tenders may follow the extended Table 6.

In the event that a reasonably stable supraventricular rhythm is restored, the patient should be taken to 60 fsw. If restoration of supraventricular rhythms occurs prior to descent to 165 fsw, the patient should be observed at 60 fsw for signs of cardiac deterioration requiring repeated defibrillation.

Adequate support of blood pressure is essential to maximize coronary and cerebral perfusion. If pressor agents are necessary, dopamine is the agent of choice. If the chamber is equipped with perforations for pressure transducers, insertion of an arterial line and a Swan-Ganz catheter may be considered. If a central line is placed in the patient for any reason, the antecubital approach is preferred over the internal jugular or subclavian

approach because of the danger of pneumothorax in the latter cases. A pneumothorax, even if not an immediate catastrophe, may cause serious problems during subsequent ascent.

A Foley catheter should be inserted unless the patient is conscious and voiding spontaneously.

The patient should be observed for signs of neurological recovery. Decadron should be administered in an attempt to minimize cerebral edema.

Post-Treatment Evaluation

Patients who have suffered dysbaric arterial air embolism should be evaluated by the following methods:

(1) neurologic examination

(2) electroencephalogram

(3) electrocardiogram and myocardial scanning

Thallium-201 accumulates maximally in normal myocardium; areas of myocardial infarction or ischemia show up as "cold spots." Technetium-99m-labeled phosphates are concentrated in infarcted and possibly in ischemic areas (Waters and Forrester, 1978). Berger, Gottschalk, and Zaret (1978) reported that the combination of both techniques is 100% sensitive in detecting myocardial infarction. These techniques should be useful in documenting the extent of myocarial injury, if present. In addition, routine use of these methods for air embolism may help clarify the natural history of this disorder.

(4) Pulmonary Evaluation. This should include: (a) a careful history which documents smoking habits, as well as prior pulmonary disease, especially asthma or recurrent pulmonary infection; (b) chest x-rays; and (c) tests of air flow. With reference to (b), in addition to standard posterior, anterior and lateral views, an expiratory film should be taken. The expiratory film may show a residual small pneumothorax or asymmetric emptying of the lungs due to

voice timbre, and dysphagia. Radiographs of the region of the neck may be helpful in indicating cases involving small amounts of subcutaneous air.

Pneumopericardium

There are no reports of clinically significant pneumopericardium in the diving literature, but evidence of this condition may be apparent radiographically.

Treatment of Mediastinal or Subcutaneous Emphysema and Pneumopericardium

There is no need for recompression in uncomplicated cases of mediastinal emphysema, subcutaneous emphysema or pneumopericardium. Breathing of 100% oxygen may hasten resorption of the air if symptoms are bothersome. In the rare event of respiratory or circulatory obstruction, recompression on oxygen to the minimal depth consistent with relief of symptoms is indicated. All cases should be monitored with serial chest films and EKGs.

Pneumothorax

Pneumothorax is a relatively uncommon phenonomen, occurring only in approximately 9-10% of cases of lung overinflation (Moses, 1964). The onset of pneumothorax is often heralded bv a sharp pain in the side during ascent.

Recompression may convert a simple pneumothorax to a tension pneumothorax by the following mechanism. During recompression, the rent in the visceral pleura remains open, allowing compressed gas to enter the pleural space. The rent may close at depth, in which case the condition remains one of simple pneumothorax as long as pressure is maintained. During ascent, however, the gas which has entered the pleural space will expand (in accordance with Boyle's Law), and the simple pneumothorax will become a tension pneumothorax. Manifestations of tension pneumothorax include cynaosis, tachypnea and hypotension during decompression.

partial bronchial obstruction not evident on plain films. Tests of air flow should routinely include forced vital capacity, FEV_1 and flows at low lung volumes. These parameters are most conveniently measured with a flow-volume loop. If the history, chest x-rays or screening tests for pulmonary function show any abnormality, pulmonary consultation to assess risk or air trapping is recommended.

SUPPLEMENTAL DISORDERS AND RELATED TREATMENT PROCEDURES

Clinical Manifestations of Extra-Alveolar Air Other Than Air Embolism

Mediastinal Emphysema

Mediastinal emphysema is usually associated with mild substernal pain, and is often described as a dull ache or a feeling of tightness, which becomes worse with deep inspiration, coughing or swallowing. Pain may radiate to the shoulders, neck or back. This condition, unless extensive, is not usually associated with shortness of breath, tachypnea or other signs of respiratory distress. Mediastinal emphysema is often accompanied by subcutaneous emphysema, and a crunching sound that is synchronous with cardiac action may be noted (Hamman's Sign). The chest x-ray is usually diagnostic.

Theoretically, it is possible for mediastinal air to be at a pressure high enough to impede cardiac filling, although this problem is rare in adults. The symptoms resemble those of cardiac tamponade, i.e., cyanosis, tachypnea, hypotension and shock. Occasionally ST-segment elevation and T-wave inversion are seen in the precordial leads. The origin and significance of these changes are uncertain. A low-grade fever and leukocytosis may occasionally be present.

Subcutaneous Emphysema

The signs and symptoms of subcutaneous emphysema include swelling and crepitation in the neck and supraclavicular fossae, sore throat, a change in

The diagnosis of pneumothorax is made on the basis of diminished or absent breathing sounds, increased percussion notes and decreased respiratory excursion on the affected side. Tracheal deviation may be present. The chest x-ray in full expiration is confirmatory. In the chamber an adequate physical exam may prove difficult and x-rays are not available. Diagnosis of pneumothorax, therefore, may not be easy.

If it is possible to exclude the possibility of cerebral air embolism by obtaining a normal neurologic history and/or examination, recompression should not be conducted because of the danger of converting a simple pneumothorax into a tension pneumothorax. If neurologic symptoms are present, however, recompression must be carried out.

The only mandatory indication for performing a pleural drainage procedure in the recompression chamber is the presence of a tension pneumothorax. Tension pneumothorax causes compression of the lung and interference with venous return, and is accompanied by severe dyspnea, cyanosis and hypotension. It is an emergency. Lesser pneumothoraces may be evacuated in the chamber depending on the skill and prior experience of the physician.

Several options are available for in-chamber management. These include the following.

(a) Insertion of a large (e.g., 10-14 gauge) angiocath through the second or third intercostal space in the midclavicular line and attachment to a flutter valve fashioned from a Penrose drain or other suitable material. This method is quick and easy and may provide very fast partial relief of a tension penumothorax. The catheter is small, however, and tends to kink. Thus, this method may not provide completely adequate decompression.

(b) An Argyle® plastic thoracostomy tube with an enclosed trocar may be used. If the operator is relatively inexperienced with tube thoracostomy the

pediatric size catheter may be tried. This catheter is relatively small and easy to place, but should be large enough to provide adequate decompression during the patient's stay in the recompression chamber. Alternatively, an adult size may be used. As with all pleural drainage procedures, the catheter should be inserted along the superior border of the rib in order to avoid the intercostal vessels. A 1 to 2-cm skin incision should be made to facilitate passage through the skin. The tube, one in place, should be secured with a skin suture and attached to a Heimlick flutter valve or to a water-sealed thoracic drainage system (e.g., Pleurovac®).

(c) The third option is to enter the pleural space by blunt puncture. When this method is used, three instruments are required: a scalpel, straight Mayo scissors and a large, curved clamp such as the Kelly clamp. The tube should be a large-bore, rigid one with multiple orifices such as the #36 Arygle®. If local anesthesia is needed for the conscious patient, careful injection of the periosteum of the superior border of the rib below the interspace used and the parietal pleura will assure a painless procedure. The patient can be "tubed" in any position, with the fifth or sixth intercostal interspace in the anterior auxillary line or the third interspace in the midclavicular line as the site of choice. If the lateral approach is used the interspace may be opened by truncal flexion or by placing the ipsilateral arm behind the head. A st ile field is prepared and the Kelly clamp placed over the end of the chest tube. Either suction, underwater seal or a Heimlick valve should be ready for connection to the tube once inserted. If nothing is available a suitable one-way valve can be fashioned by placing a Penrose drain over the end of the tube. Once preparation is complete, a 2-cm skin incision is made one interspace below the site of entry. Using the closed tips of the Mayo scissors the skin incision is pushed up one interspace and the patient is

essentially stabbed over the superior aspect of the lower rib at the site of entry. In adult males, near-maximal force may be necessary to traverse both the intercostal musculature and the parietal pleura in one swift motion. A successful effort is marked by the hiss of air either leaving the interpleural space (tension pneumothorax) or entering the interpleural space (as the lung completely collapses). The handles of the scissors are then spread so that the tips are 3-4 cm apart, and maintaining this separation, the Mayo scissors are forcefully withdrawn from the chest, leaving a single hole from skin to lung large enough to admit a tube. Before placing the tube it is essential to put the gloved finger into the interpleural space to certify three things:

1) the interpleural space has been reached

2) the lung is not adhesed to the chest wall

3) the hole is large enough and easily probed

The chest tube is then inserted, mounted on the Kelly clamp by using the curve of the clamp to direct the tube to the apex of the lung and suction is applied. The tube may be sutured in place using 1-0 silk in a generous U-stitch. A final check for tube placement and patency is the vapor that mists on the tube wall as suction is applied, and the respiratory excursion of the water column once the lung is inflated.

The following precautions should be noted.

1. Conservative methods involving dissection and multiple incisions are the source of false passages and incorrect tube placement.

2. Even with this blunt technique, a laceration of the intercostal artery, which runs on the inferior surface of the rib, may lead to significant bleeding and require surgical repair.

3. Without respiratory excursions in the underwater seal, one cannot prove that the tube is functional; hence, a recurrent pneumothorax may result.

4. Too large a skin incision or intercostal dissection may leave the patient with a sucking chest wound despite the presence of the tube.

5. Persistent bubbling of the underwater seal or air leaking though the Heimlick valve means a bronchopleural fistula has occurred, unless a source of the air can be found at the insertion site.

6. Never clamp a chest tube during ascent. A clamped or otherwise occluded tube may allow re-expansion of residual intrapleural air during ascent, causing a recurrence of tension pneumothorax.

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Royal Navy Treatment Table 71: Modified Air Recompression Therapy*

Gauge Depth (metres)	Stoppages/ Ascent [hours (h) and minutes (min)]	Elapsed Time (hours and minutes)	Rate of Ascent (metres/hour)
70	30 min	0000-0030	—
70-63	7 min	0030-0037	60 m/h
63-51	2 h	0037-0237	6 m/h
51-39	4 h	0237-0637	3 m/h
39-29	5 h	0637-1137	2 m/h
29-20	6 h	1137-1737	1 5 m/h
20-10	10 h	1737-2737	1 m/h
10-0	20 h	2737-4737	0 5 m/h
Surface		4737	

Royal Navy Treatment Table 72: Modified Air Recompression Therapy

Gauge Depth (metres)	Stoppages/ Ascent [hours (h) and minutes (min)]	Elapsed Time (hours and minutes)	Rate of Ascent (metres/hour)
50	2 h†	0000-0200	—
50-39	3 h 40 min	0200-0540	3 m/h
39-29	5 h	0540-1040	2 m/h
29-20	6 h	1040-1640	1 5 m/h
20-10	10 h	1640-2640	1 m/h
10-0	20 h	2640-4640	0 5 m/h
Surface		4640	

* Ministry of Defence (Navy). Diving Manual B.R. 2806. London: Her Majesty's Stationary Office, 1972, p 5-49.

† The period of two hours can be reduced and decompression started earlier if the patient's symptoms have cleared.